Dissertation

"STUDY ON INFLUENCE OF LRINEC SCORING SYSTEM (LAB RISK FACTORS INFLUENCING NECROTIZING FASCIITIS), LIMB VASCULARITY AND MICROBIAL FLORA ON OUTCOME OF NECROTIZING FASCIITIS"

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CERTIFICATE

This is to certify that, the dissertation entitled **"**STUDY ON INFLUENCE OF LRINEC SCORING SYSTEM (LAB RISK FACTORS INFLUENCING NECROTIZING FASCIITIS), LIMB VASCULARITY AND MICROBIAL FLORA ON OUTCOME OF NECROTIZING FASCIITIS**"** is the bonafide work done by **Dr.KALYAN KUMAR M.S** during his MS (General Surgery) course 2012-2015, done under my supervision and is submitted in partial fulfillment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamilnadu Dr.MGR Medical University, April 2015 examination.

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DECLARATION

I, certainly declare that this dissertation titled **"STUDY ON INFLUENCE OF LRINEC SCORING SYSTEM (LAB RISK FACTORS INFLUENCING NECROTIZING FASCIITIS), LIMB VASCULARITY AND MICROBIAL FLORA ON OUTCOME OF NECROTIZING FASCIITIS"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The TamilNadu Dr. M.G.R Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Master of Surgery Degree Branch I (General Surgery).

Date:

Place: **Dr. KALYAN KUMAR M.S**

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As I walk down the memory lane I realize with a deep sense of humility that what I have done now would not have materialized, but for certain luminaries, who have enlightened my path to wisdom.

"Surgery is learnt by apprenticeship and not from textbooks, not even from one profusely illustrated" – Ian Aird.

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What good is a potter without his clay and what good is a study without the active participation of the patients. My heartfelt thanks go to each and every patient who agreed to be a part of this study and also my apologies to them in case of any inconvenience caused.

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INTRODUCTION

INTRODUCTION

Necrotizing fasciitis is a rapidly progressive infection primarily involving the fascia and the subcutaneous tissue. Initially the overlying skin is relatively normal but the necrotic process proceeds underneath.

The patient becomes extremely toxic and later the skin becomes painful, red and necrotic as it is deprived of its blood supply

The fascial necrosis is usually wider than the skin involvement that is visible clinically.

Pathophysiologically, it is a septic thrombosis of the vessels between the skin and the deep layers.

Assessment of various risk factors of necrotizing fasciitis helps in bringing down the morbidity and mortality of necrotizing fasciitis.

In this study a conscious attempt is made to correlate LRINEC scoring system, microbial flora and Doppler assessment of the affected limb with the outcome and prognosis of necrotizing fasciitis.

AIMS AND OBJECTIVES

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To study the influence of the following factors on the outcome of patients with Necrotizing Fasciitis :

1. LRINEC (LAB RISK FACTORS INFLUENCING NECROTIZING FASCIITIS) scoring system
2. Doppler study of affected limb
3. Microbial flora

Above factors were analysed in this study to predict the outcome of necrotizing fasciitis which has got a high degree of morbidity and significant mortality so that the analysis would be helpful in bringing down the morbidity of the disease.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Wilson first introduced the term Necrotizing Fasciitis in 1952 and it is the preferred term today describing the most consistent and key feature of this disease that is fascia necrosis.[3]

Necrotizing fasciitis is an acute, life threatening infection of the superficial fascia and subcutaneous tissue caused by a variety of aerobic and anaerobic bacteria. The clinical process at first appears to be a low grade cellulitis, but fulminant infection develops rapidly in subcutaneous fascia, which may become liquefied with accompanying fat necrosis, thrombosis of subcutaneous vessels and occasional myositis and myonecrosis. As the blood supply to the skin is compromised, cutaneous erythema and oedema progress to cyanosis, bullae and gangrene. Cutaneous gangrene is associated with fever, shock and higher mortality rate.[7]

Necrotizing fasciitis tends to occur in diabetics, alcoholics, intravenous drug abusers, immunocompromised patients, and as a post-operative complication.

A patient with necrotizing fasciitis usually presents with the clinical features of cellulitis, including erythema , swelling and local

heat in the affected area. There is pain in the area concerned, that is out of proportion to the severity of cellulitis[45].

Systemic features of toxicity, including fever, tachycardia and leucocytosis, are also out of proportion to the apparent severity. If left untreated the skin becomes shiny, hot and exquisitely tender but discrete margins do not develop. The skin forms blisters and bullae that contain clear thin haemorrhagic fluid.[8]

Soft tissue gas is an uncommon feature of necrotizing fasciitis but is seen when there is anaerobic infection. The diagnosis is difficult and rests on high index of suspicion in the clinical settings stated above.[8]

Radiological examination in the form of plain radiograph or computed tomographic scan may show gas in the tissues. The diagnostic test is a full thickness biopsy of the affected area or surgical exploration.[8]

Although initiation of antimicrobial therapy is essential, these medications do not stop the progress of necrosis initiated by the organism released toxins. Surgical intervention in the form of debridement or amputation is essential.[9]

Aggressive debridement of infected tissue is the only treatment option, which often leaves the patient with an extensive post operative wound.[10]

Post operative care requires the patient to receive the appropriate intravenous antibiotics, an effective wound management regimen and adequate nutritional support.[10]

A functional extremity can usually be salvaged in fasciitis, if not, amputation can be safely performed later. Immediate amputation is necessary when there is diffuse myositis with complete loss of blood supply or when adequate debridement will leave a useless limb. When the viability of the remaining tissue is assured and the infection has been controlled , soft tissue deficits can be covered with skin grafts.[11]

HISTORICAL ASPECTS

1. In the 5th century BC , Hippocrates was the one who described Necrotizing Fasciitis as complication of erysipelas.[12]

2. In 18th and 19th centuries, the British naval surgeons referred to Necrotizing Fasciitis as hospital gangrene.

3. In USA, the first report of hospital gangrene was by a Confederate Army surgeon Joseph Jones during the Civil War.[14]

4. Wilson proposed the term necrotizing fasciitis in 1952, reminiscing the key feature of the disease as fascial necrosis.[3]

BRIEF SURGICAL ANATOMY OF SKIN AND SUBCUTANEOUS TISSUES

Skin consists of two components – Epidermis and Dermis.

The surface epithelium of the skin is the **Epidermis** and is of keratinized stratified squamous variety.[15]

The various skin appendages – sweat gland , sebaceous glands , hair and nails – are specialized derivatives of this epidermis , which is ecdodermal in origin.

The deeper **Dermis** is mesodermal in origin and consists mainly of collagen fibres together with some elastic tissue, blood vessels , lymphatics and nerve fibres.

The skin is connected to the underlying bone , muscles or deep fascia by a loose areolar connective tissue , this layer is referred to as **superficial fascia** , it is of variable thickness and fat content.

The limbs and body are wrapped in a membrane of fibrous tissue - the deep fascia.[16]

The **Deep fascia** is a dense , organized connective tissue layer , devoid of fat , that covers most of the body parallel to the skin and subcutaneous tissue (superficial fascia) . Extensions from its inner

surface invests deeper structures, such as individual muscles and neurovascular bundles , as the **investing fascia**.

Beneath deep fascia are the muscles , the bones , the joints with synovial sheaths and the cavities (eg:peritoneal)

WOUND HEALING

A clear understanding of healing is vital to a rational approach to the practice of surgery.

There are three phases of wound healing –

1. Inflammatory phase ,
2. Proliferative phase and
3. Remodelling phase.

Inflammatory phase :

Inflammatory phase begins immediately after wounding and lasts 2 to 3 day .Wounding is immediately followed by coagulation, altered vascularity, and inflammation, all of which modulate wound healing. Coagulation is mediated by platelets, and during thrombus formation, platelet factors that enhance fibroblast migration and proliferation are released.[17]

The normal inflammatory response soon follows as small blood vessels dilate, capillary permeability increases, and peripheral neutrophils and then monocytes migrate into the wound. As monocytes ingest material , they are transformed into macrophages that phagocytize debris as well as enzymatically. destroy bacteria. Macrophages also play a role in the induction of collagen synthesis.[18,19]

Prostaglandins also play a significant role in this process.[20]

Proliferative phase : (3rd day to 3rd week)

Proliferative phase consists mainly of fibroblast activity, production of collagen, growth of new blood vessels as capillary loops, and re-epithelisation of the wound surface.[23]

Collagen provides strength and stability for all tissues of the body. The strength and integrity of all tissue repairs relies on the cross linking and deposition of collagen[38].

It is not the collagen synthesis but the collagen cross linking that is the bottom line for the surgeon because it is cross linking that provides strength and integrity to any repair.

Collagen degradation , mediated by enzyme collagenase, is equally important as collagen synthesis in wound repair. In normal unwounded dermis collagen synthesis and degradation occur in equilibrium. After wounding, however , the rates of collagen synthesis and degradation rise and fall in an ordered, sequential fashion , so that enough collagen is synthesized, cross linked, deposited, and removed to provide wound strength and integrity without excessive scarring.[21]

Remodelling phase :

It is characterized by maturation of collagen type (type 1 replacing type 3 in the ratio of 4:1).

The myofibroblasts (a fibroblast-like cell with smooth muscle components) are the cells responsible for wound contraction.

It is commonly hypothesized that myofibroblasts are the responsible contractile cells and that it is the collagen that holds the newly contracted tissues in position.

Types of wound healing

1. Healing by Primary intention
2. Healing by secondary intention

3. Delayed primary closure

Healing by Primary intention

Healing by primary intention occurs when full thickness wound edges are approximated shortly after the primary wound has been created. Epithelisation and contraction have little to do with the healing by primary closed wounds, even though minimal epithelisation occurs within 24 hours and seals the wound from bacterial contamination.

Healing by secondary intention

This is healing by natural biological processes without surgical intervention, which usually occurs in large wounds associated with skin and soft tissue loss. Although epithelization and collagen deposition are involved, contraction is the most important phenomenon in the spontaneous closure of large open wounds. Unless contraction occurs and brings dermal structures together, the granulating surface is covered only by a layer of epithelial cells that are useless in providing any coverage with strength and integrity.

Delayed primary closure

Closure of grossly contaminated incisions/wounds should be delayed, allowing time for host inflammatory and immune responses to control contamination. Most significant is that delayed primary closure does not delay the development of wound strength.

AETIOLOGY

Necrotizing infections of soft tissues are infections by virulent bacteria that have the ability , usually by the production of toxins , to cause widespread necrosis.

The soft tissues can be subcutaneous (eg : necrotizing fasciitis) , muscle (eg : gas gangrene) and less frequently skin.[8]

Soft tissue infections produce progressive tissue destruction which eventually progress further to soft tissue and limb loss and finally mortality.

Blood supply to the fascia is more tenuous than that of muscle or skin and thus the fascia is more vulnerable to infectious processes.[32]

Necrotizing fasciitis is defined by Bisno as : 'Necrotizing fasciitis is deep seated infection of subcutaneous tissue that leads to progressive destruction of the fascia and fat.[8]

The incidence of Necrotizing fasciitis has been reported to be 0.40 cases per 1lakh population.[24]

Though Necrotizing Fasciitis is rare, certain conditions predispose patients to developing the disease, including immunocompromised states such as diabetes mellitus, malignancy, AIDS to name a few and also those with intravenous drug or alcohol problems.[24]

Necrotizing fasciitis may occur following trauma, burns and lacerations. Even minor trauma, insect bites and needle sticks, may cause NF. Patients with peripheral vascular disease and atherosclerosis also have an increased risk.

Summarising, entrance of bacteria can occur from any break in the integrity of skin and can occur in patients with preexisting skin conditions such as boils, psoriasis, pressure ulcers, or perianal abscesses.

CLASSIFICATION

Necrotizing fasciitis is classified depending on type of organisms cultured [25,26,27]

1. Type 1 NF - polymicrobial infection (aerobic and anaerobic bacteria such as *Clostridium* and *Bacteroides* species).

2. Type 2 NF - group A *Streptococcus : (S. pyogenes*) with or without a coexisting *Staphylococcal* infection.

3. Type 3 NF is associated with Vibrio vulnificus, which enters the subcutaneous tissues via puncture wounds from fish or marine insects.

Necrotizing fasciitis classification

Type 1	Type 2
Bacteroides , Eikenella	Group A *Streptococcus*
Peptococcus , Pseudomonas	+/-*Staphylococcus*
Fusobacterium ,Candida	
Clostridium , Cryptococcus	
Corynebacterium, Histoplasma	
Streptococcus (not group A) , *Vibrio*	
Escherichia , Staphylococcus	
Enterobacter , Shigella	
Proteus , Neisseria	
Klebsiella , Pasturella	

Clinically, based on the length and extent of the disease, Necrotizing fasciitis can be divided into 3 groups ,

1. ***Fulminant NF***—rapid disease progression and are often present in shock.They have typically had symptoms for only hours.
2. A***cute NF***—have symptoms for days. In most cases, large areas of their skin are involved.

3. S***ub-acute NF***—have symptoms for weeks. localized area of skin is generally affected.

Necrotizing fasciitis can affect any area of the body, but it most commonly occurs on the extremities.

MYONECROSIS

Bacterial myonecrosis is the preferred term when muscle invasion occurs.

It is an uncommon disease with a grave prognosis even with aggressive therapy.

- Non clostridial myonecrosis is caused by the same organisms as is necrotizing fasciitis and the initial symptoms may be identical.[47]

Chronic skin ulcers and perineal infections were the most frequent primary sites. Soft tissue gas was usually present.

Non clostridial myonecrosis is caused by a group of bacteria , the most predominant of which are the anaerobic streptococci.[8]

If not treated it progresses to gangrene , toxaemia and shock.

- Clostridial myonecrosis is caused by clostridium perfringens , clostridium novyi , and clostridium septicum.[48]

PATHOPHYSIOLOGY

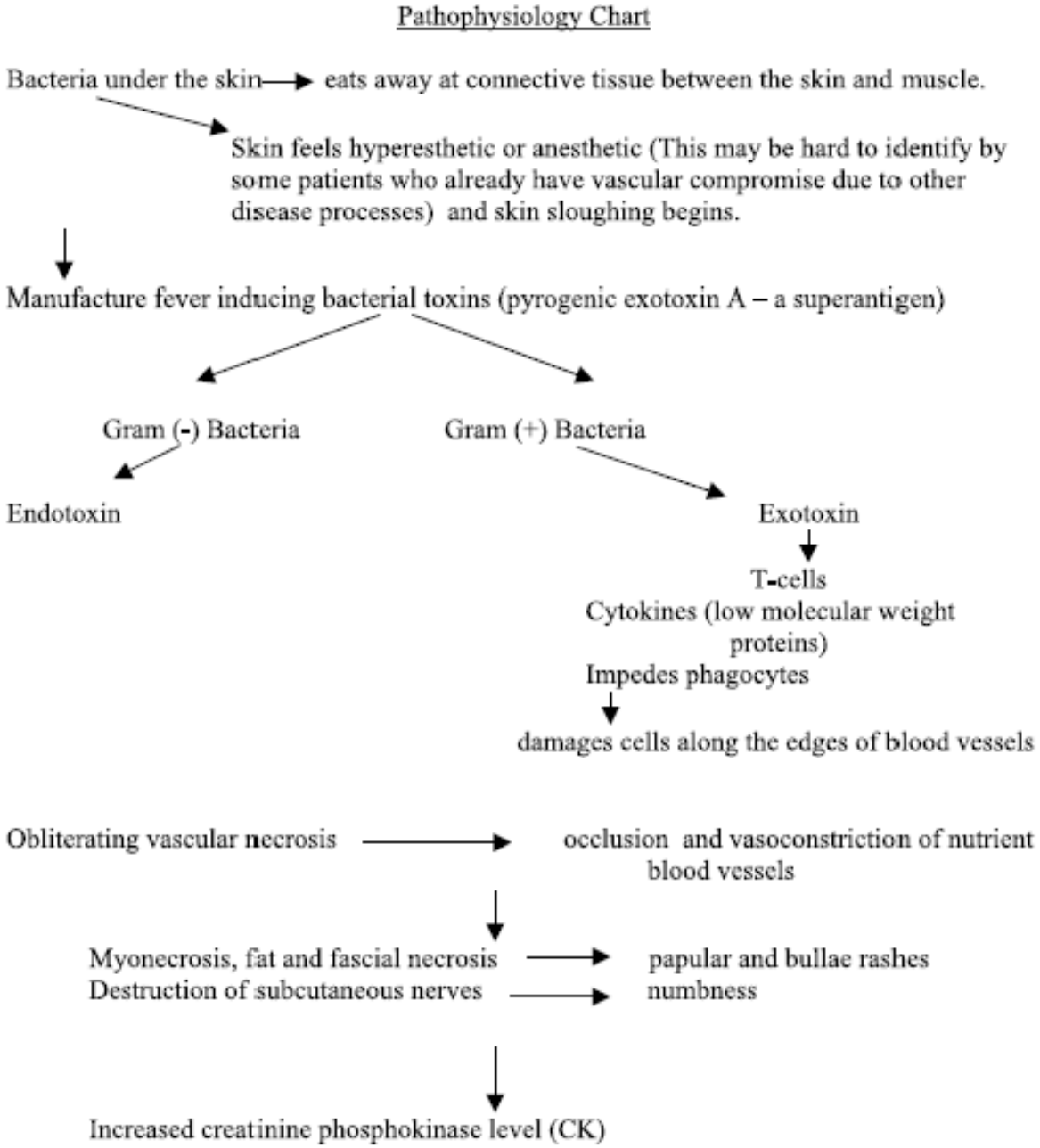
In necrotising fasciitis, bacteria spread through subcutaneous layer directly from trivial break in the skin or from distant places of infection, for example, intestinal fistulae, kidney stones or streptococcal pharyngitis.[14]

Host factors responsible for weakening resistance to bacteria, such as a compromise in the integrity of skin or mucosa, immunosuppression and hyperglycaemia, facilitate bacterial growth.[33]

Bacterial toxins facilitate the spread of pathogen and aid in tissue necrosis. Necrosis of soft tissues progresses as fast as 1 inch an hour, which may not be easily recognizable early because the necrosis of subcutaneous tissue and fascia is far more extensive than skin.[34]

Blister or bulla formation is important diagnosis of NF because they are rare in cellulitis and erysipelas. They are caused by ischemia in the vessels supplying the skin as a result of vessel necrolysis and thrombosis. Rarely, lymphangitis, lymphadenitis, and venous thrombosis are seen.[14]

Patients frequently succumb to NF due to septicemia and multiple organ failure.



CLINICAL FEATURES

Pain , warmth, swelling are the three predominant symptoms of necrotizing fasciitis. But these are not specific to the disease and also are not present in all cases.[42]

There are certain hard clinical signs that are very specific to necrotizing fasciitis but these occur later in the course[4].

These include –

1. bullae
2. ecchymosis of the skin followed by skin necrosis
3. gas in tissues by clinical examination or radiological examination
4. numbness

These findings are strongly indicative of necrotizing infections and prompt immediate surgical exploration .But these signs are present only in a minority of cases.

Other less specific clinical signs are-

1. Pain disproportionate to the disease
2. Oedema extending beyond skin erythema

3. toxicity

4. Progression of disease despite aggressive antibiotic therapy

Clinical features of necrotizing fasciitis as the disease progress through clinical stages

Stage 1 (Early)	**Stage 2 (intermediate)**	**Stage 3 (late)**
Tenderness to palpation (extending beyond the apparent area of skin involvement) Erythema Swelling Warm to palpation	Blister or bullae formation(serous fluid) Skin fluctuance Skin induration	Hemorrhagic bullae Skin anesthesia Crepitus Skin necrosis with dusky discoloration progressing to frank gangrene

Necrotizing Fasciitis presenting with blebs and blisters

Systemic manifestations of necrotizing fasciitis

Toxic appearance	Neuralgia
Fever	Weakness/fatigue
Chills	Tachycardia
Constitutional symptoms	Tachypnea
Shock	Decreased urinary output
Multiorgan system failure	Death
Mental status changes	

INVESTIGATIONS

1. Routine Blood Investigations -

Haemoglobin – it is a useful investigation to know the general status of the patient and to know about the fitness of the patient for distinctive operative procedures. Reduced haemoglobin may add in addition to the other causes of non healing of wound.

Total WBC count - this includes the count of polymorphs, lymphocytes and eosinophils[14].

Differential WBC count – A raise in polymorphs count will give a clue to the underlying infection and severity of infection.

ESR – it s increased in long standing diseases like tuberculosis etc

CRP – it's elevated[14]

Bleeding time and clotting time – altered levels may require correction when contemplating any surgery for the patient[18]

Fasting blood sugar – to know the presence or absence of diabetes and to assess the degree of control of diabetes[21].

Serum creatinine – it s the more sensitive indicator of renal function, which may be hampered in renal failure as a result of long standing diabetes mellitus or as a result of acute necrotizing fasciitis going for renal failure.

Blood urea – also indicates renal function , but may vary with hydration of the patient.

HIV 1 & 2 – for diagnosis of underlying immunodeficiency syndrome.

HBsAg – for diagnosis of underlying hepatitis B infection.

2. Examination of the urine -

For sugar – detection of glucose levels in urine to rule out diabetes mellitus

For ketone bodies – to rule out complication of diabetes – diabetic ketoacidosis.

3. Bacteriological examination pus c/s –

Examination of the discharge for culture and sensitivity is important. A baseline bacterial culture with sensitivity result is useful. It provides a guideline for starting chemotherapy[45].

LRINEC Scoring System

(Lab Risk Indicators of Necrotising Fasciitis)

Early operative debridement is a major factor determining the outcome of necrotizing fasciitis. But early recognition is difficult clinically. This scoring system is for distinguishing necrotizing fasciitis from other soft tissue infections based on laboratory tests performed for the evaluation of severe soft tissue infections. The following parameters are taken into account

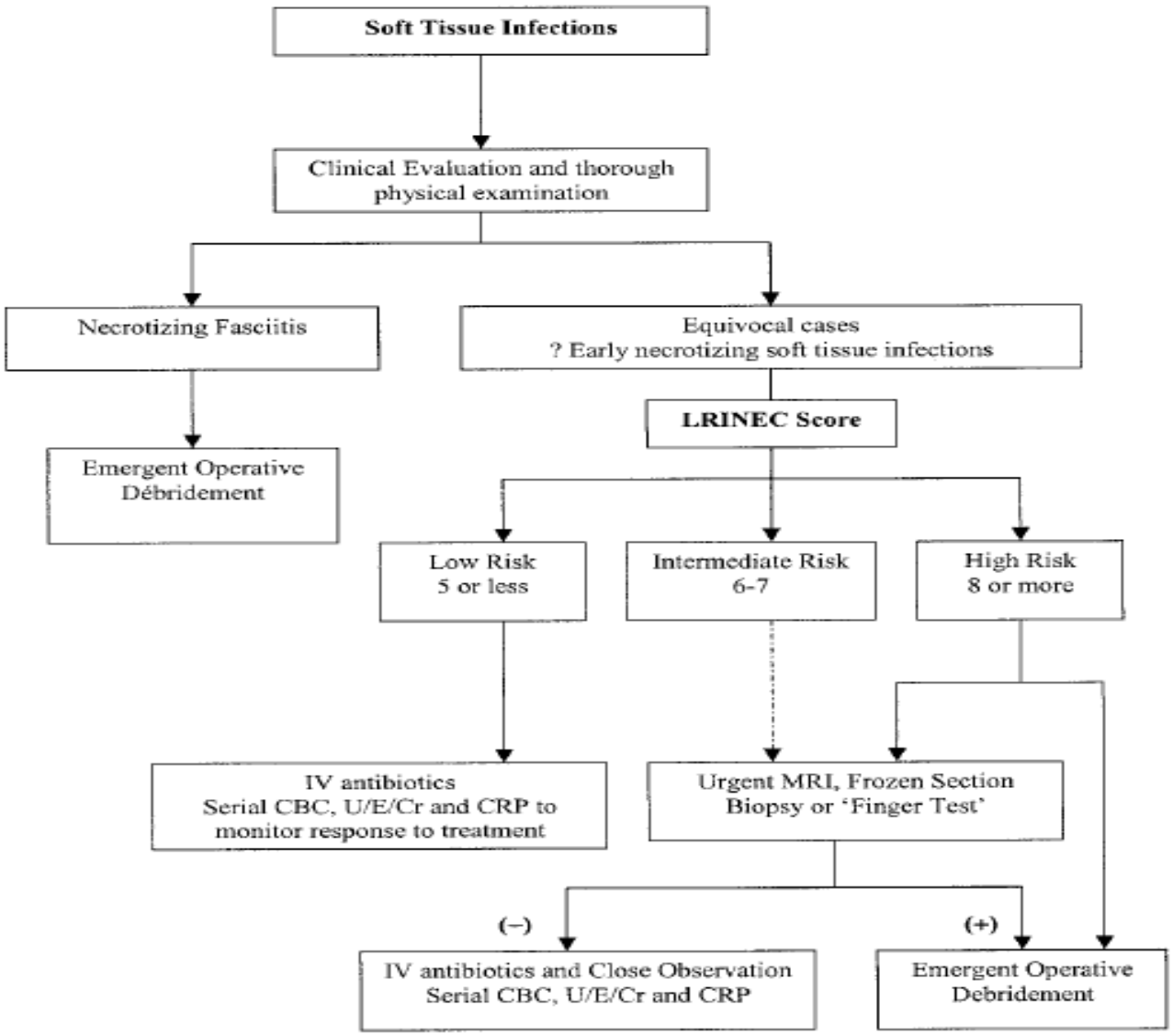
1. C-reactive protein
2. Total white cell count,
3. haemoglobin,
4. sodium,
5. creatinine
6. glucose

The LRINEC score is a robust score capable of detecting even clinically early cases of necrotizing fasciitis. The variables used are routinely measured to assess severe soft tissue infections. Patients with a LRINEC score of >6 should be carefully evaluated for the presence of necrotizing fasciitis[7]

- maximum score - 13;
- score of >/6 - raise the suspicion of necrotizing fasciitis and
- score of >/8 - strongly predictive of this disease

The LRINEC (laboratory risk indicator for necrotizing fasciitis) score

Variable	Score
C-reactive protein (mg/l)	
<150	0
150 or more	4
Total white cell count (per mm^3)	
<15	0
15–25	1
>25	2
Hemoglobin (g/dl)	
>13.5	0
11–13.5	1
<11	2
Sodium (mmol/l)	
135 or more	0
<135	2
Creatinine (μmol/l)	
141 or less	0
>41	2
Glucose (mmol/l)	
10 or less	0
>10	1



Radiographic studies -

Soft tissue gas is a uncommon feature of necrotizing fasciitis but is seen when there is anaerobic infection. This can be visualized on plain x rays of the involved area.

DUPLEX DOPPLER ULTRASOUND -

'Duplex Doppler ultrasound uses standard ultrasound methods to produce a picture of a blood vessel and surrounding organs. A computer converts the Doppler sounds into a graph that provides information about the speed and direction of blood flow through the blood vessel being evaluated. With this type of Doppler ultrasound, it is possible to see the structures inside the body (2D) and evaluate blood flow (pulsed wave) within those structures at the same time.

To do this, the ultrasound machine uses the 2 methods of ultrasound simultaneously. Blood flow in individual blood vessels is most commonly evaluated by duplex Doppler ultrasound. Doppler ultrasound is used to evaluate blood flow in legs and in identifying blockages in arteries'[56]

MANAGEMENT

Successful management of necrotizing fasciitis includes –

1. Early diagnosis
2. Surgical debridement
3. Amputation of extremity
4. Wound care
5. Antimicrobial therapy
6. Intensive supportive care
7. Hyperbaric oxygen

Surgical debridement

Adequate surgical debridement is essential to the successful management of necrotizing fasciitis. This will require radical excision of all necrotic tissue, drainage of involved fascial planes, and extensive fasciotomy.[8]

Careful revaluation of the wound and formal re-exploration in the operating theatre under general anaesthesia is also required, often on two or three further occasions.[8]

Surgical debridement is a form of mechanical debridement. Mechanical debridement, including sharp debridement, wet to dry dressings and high pressure irrigation or pulsed lavage are well accepted therapeutic measures.

Thorough sharp debridement of all non viable soft tissue and bone from the open wound is accomplished mainly with a scalpel, tissue nippers, and or curettes.[44]

Autolytic debridement occurs naturally in a healthy moist wound environment with maintained arterial perfusion and venous drainage.

Enzymatic debridement (using topical, proteolytic enzymes such as collagenase) however, is commonly used as an adjunctive therapy in the management of wounds.

Wound management

Generally, a moist wound environment bandaged to protect it from trauma and local contamination has been shown to facilitate the healing process. The type of dressing depends upon factors such as size, depth, location, and the wound surface[52]

Dressings_:

The types of dressings can be broadly divided into

films,

composites,

hydrogels,

hydrocolloids,

alginates,

foam and

other absorptive dressings including NPWT-Negative Pressure Wound Therapy.[43]

The choice of one over the other is made by considering the wound characteristics and treatment goals.

TOPICAL THERAPIES/AGENTS

Saline / amorphous hydrogels: skin cleansers	Clean / infected wounds	Undefined
Detergents / antiseptics	Contaminated or infected wounds	Healthy granulating wounds

Topical antibiotics silver sulfadiazine,	Contaminated or infected wounds	Healthy granulating wounds
Enzymes :collagenase, papain, urease etc	Necrotic/escharotic wounds	Healthy or infected wounds
Growth factors – becaplermin gel,	Neuropathic diabetic foot ulcers	Infected / necrotic wounds
Dermal skin substitute- Apligraf,	Diabetic ulcers , Venous stasis ulcers	Infected / necrotic wounds

***NEGATIVE PRESSURE WOUND THERAPY* : (NPWT)**

Negative pressure wound therapy or vacuum assisted wound closure consists of the use of a porous sponge within the wound, covered by a airtight occlusive dressing, to which a vacuum is applied.[61]

HYPERBARIC OXYGEN:

The use of hyerbaric oxygen (HBO) raises the dissolved oxygen saturation in plasma from 0.3% to nearly 7% . this rise in oxygen saturation increases the interstitial diffusion distance of oxygen four to fivefold.[43]The broadening use of transcutaneous oximetry has permitted evaluation of patients that will likely benefit from HBO.[41]

ANTIBIOTICS :

Use of broad-spectrum antibiotics is needed particularly in type 1 NF, until culture and sensitivity results are available. Numerous regimens are employed; a commonly used one is combining penicillin for gram positive cocci, aminoglycoside for gram negative aerobes, a third-generation cephalosporin, and metronidazole for anaerobes.[13,43]

Vancomycin is added in patients with suspected methicillin-resistant S aureus or with penicillin allergy. In immunocompromised patients, coverage of Pseudomonas is essential.

Dermagraft

This is a wound healing product which consists of neonatal dermal fibroblasts cultured in vitro on a bioabsorbable polyglactin mesh by tissue engineering technique. This graftskin or dermagraft (available as Apligraft) is an allogenic bilayered, metabolically active cultured skin equivalent , which has upper epidermal and a lower dermal layer and contains human skin cells.[50]

PLATELET DERIVED GROWTH FACTOR (BECAPLERMIN)

Becaplerin [recombinant human platelet derived growth factor – BB (rh PDGF-BBI)] is quickly emerged as one of the leading candidates for clinical trials.It is available in united states as Regranex(Becaplermin) gel

for treatment of chronic wounds which helps in complete healing.[52]

GLYCAEMIC CONTROL

The control of glucose levels should be as strict as possible, and blood glucose levels above 10mmol/L must be avoided, as they are associated with impaired function of the leucocytes, both polymorphonuclear and mononuclear cells. This degree of glucose control should not be achieved by excessive restriction of food intake in someone with tissues to heal and an infection to combat. Insulin will often be required in those not previously receiving it, even if only temporary.

SMOKING

The patient should abstain from smoking because of its ill effects on the peripheral circulation during wound healing process. Patient must

be motivated to strictly adhere to the abstinence for better response to treatment.

AMPUTATION

1. Amputation is indicated for infections not responding to higher antibiotics and surgical debridement.

2. Open amputation is usually done and is performed by any of the two methods.[18,21]

3. Guillotine amputation is performed initially and later revision to a proximal level can be planned after the infection is brought under control.

4. Or else an open amputation may be performed at the definitive level by initially inverting the flaps and then packing the wound.secondary closure can be planned 10 to 14 days later.[19]

Complications of amputation:

1. Hematoma
2. Infection
3. Wound necrosis

4. Contractures

5. Pain

Amputations of lower extremity:

1. *Transtibial amputations:*

The importance of preserving the knee joint in the successful rehabilitation of a patient with a lower extremity amputation cannot be over emphasized.

Transibial amputation is divided into 3 levels. The appropriate level must be determined for each individual patient.

The ideal bone length for a below knee amputation stump is 12.5 to 17.5 cms, depending on body height.(rule of thumbs 2.5 for each 30 cm height)[55]

Ideal/most satisfactory level is about 15cms distal to medial tibial articular surface.A stump less than 12.5 cm is less sufficient.

2. *Disarticulation of knee:*

1. It results in an excellent end bearing stump.

2. Its use in elderly is limited.

3.Transfemoral amputations:

Above knee amputations are classified as short transfemoral, medial transfemoral, log transfemoral and supracondylar.

Amputation through thigh is second in frequency to only to transtibial amputation.

Here patients knee joint is lost. So it is extremely important for the stump to be as long as possible to provide a strong lever arm for control of the prosthesis.

4.Disarticulation of the hip:

Hip disarticulation occasionally is indicated after massive trauma, for arterial insufficiency, for infection like necrotizing fasciitis or for certain congenital limb deficiencies.[56]

5.Hemipelvectomy:

Most often performed far tumors that cannot be adequately resected by limb sparing techniques or hip disarticulation. Other indications are life threatening infection/arterial insufficiency.[56]

MATERIALS AND METHODS

MATERIALS AND METHODS

The data for the study was obtained from patients hospitalized with a provisional diagnosis of necrotizing fasciitis on clinical evaluation and who are admitted at Madras Medical College (MMC) and Rajiv Gandhi Govt. General Hospital (RGGGH). Patients presenting with signs and symptoms of Necrotizing Fasciitis admitted during August 2013 to August 2014 at Madras Medical College (MMC) and Rajiv Gandhi Govt. General Hospital (RGGGH), were counseled for investigation and treatment of Necrotizing Fasciitis and its complication. 50 patients were treated in the above hospital over the period of 1 year.

Exclusion criteria:

- Very sick patients,
- age more than 65 years,
- already established cases of DVT

Assessment of parameters:

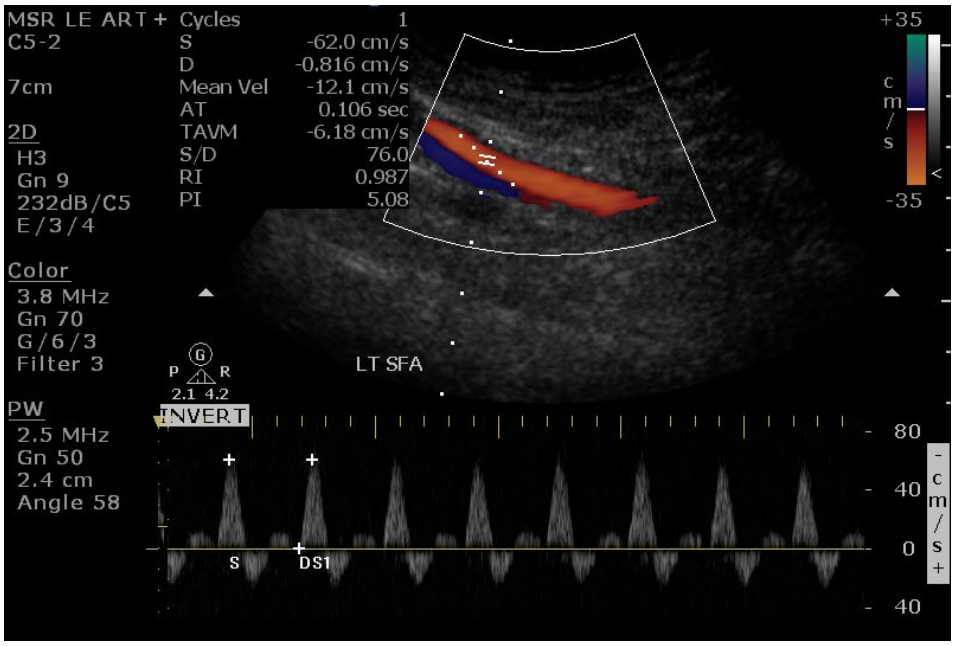
All consenting patients with necrotizing fasciitis would be clinically examined after history taking and then subjected to blood investigations as follows :

- C-reactive protein
- Total white cell count
- Haemoglobin
- Sodium
- Creatinine
- Glucose
- Wound c/s

And then Doppler ultrasound of the affected limb would be taken to assess the vascularity of the limb

Hand held doppler

NORMAL VELOCITY AND SPECTRAL PATTERN ON DOPPLER



Name, age, occupation, socioeconomic status, residence were recorded. The presenting complaints and details were recorded in chronological order.

Detailed physical examination including nutritional status, built, status of vascular system and neurological system were recorded. Detailed local examination of involved part done .

On admission , general and medical treatment of necrotizing fasciitis was done and followed by wound debridement as the definitive procedure . The patients were later managed by regular wound dressings , antibiotics , and supportive therapy for maintenance of blood pressure and renal status and in few cases vacuum assisted dressings were tried for faster healing . Once the wound was healthy split skin grafting and secondary suturing was done in most cases . Some cases healed by secondary intention . Some cases had to undergo major amputations for control of infection and its spread .

Diabetic patients were managed by diabetic treatment like diabetic diet, sugar restriction and anti diabetic treatment was given with oral hypoglycaemic drugs and insulin.

Patients who developed renal complications were managed by salt restrictions , dialysis and supportive renal treatment.

Supportive treatment was given for patients who had bed sores as a complication of NF by regular dressings and water beds .

Patients who went into septicaemia were managed in intensive care units on ventilators under guidance of anaesthetists and physicians.

Post discharge patients were followed up to one month regularly on out patient basis for dressings , further management of diabetes and hypertension and also to review liver and renal parameters.

Major amputation patients were advised for clutches and artificial prosthesis 4 weeks after surgery.

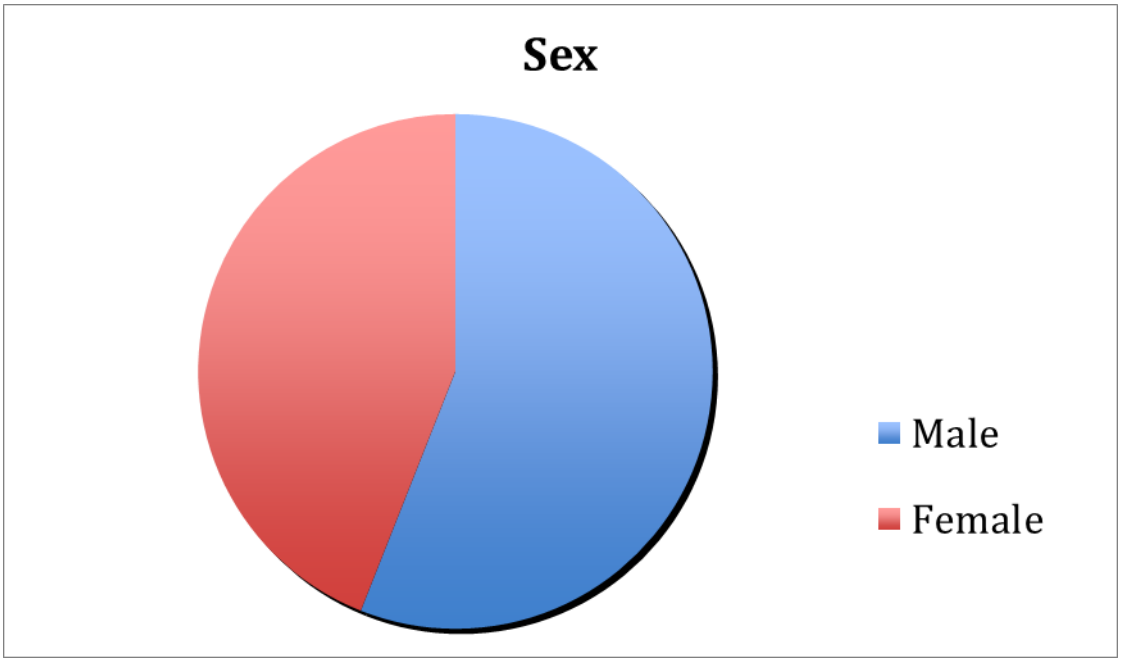
RESULTS

Results

The following are the results of our study taking into account the various factors that possibly affect the outcome of necrotising fasciitis and its morbidity.

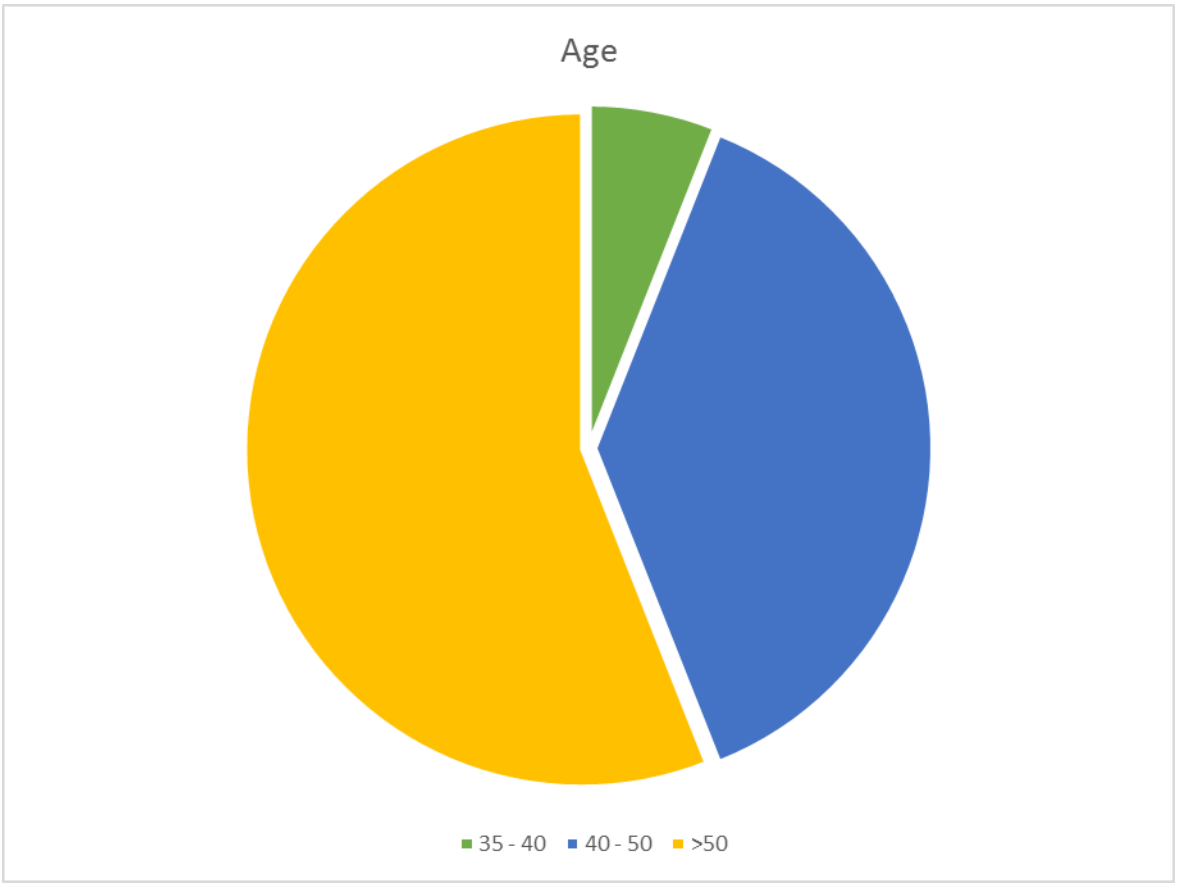
Sex

SEX	NO OF PATIENTS
MALE	28
FEMALE	22



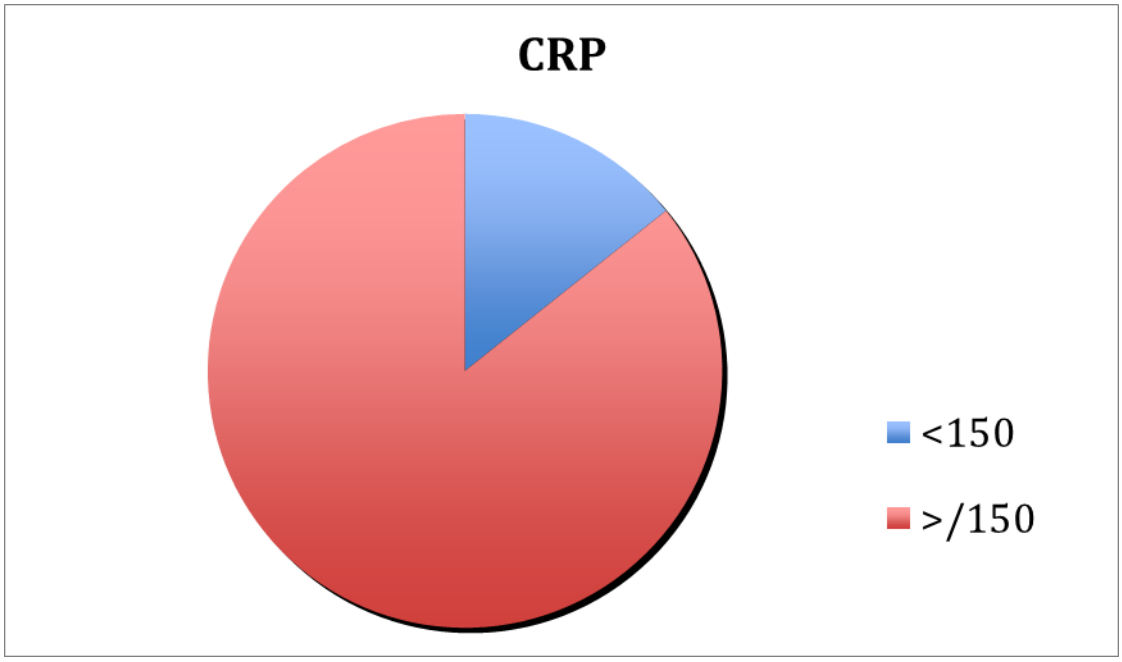
Age

Age	NO OF PATIENTS
35 – 40	3
40 – 50	19
>50	28



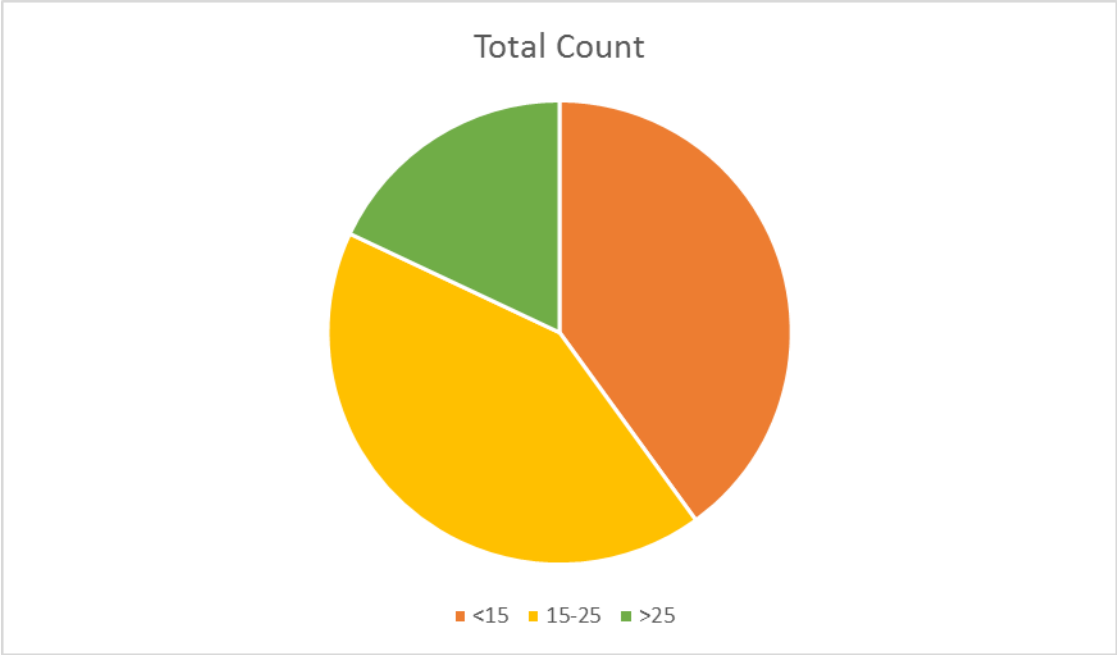
CRP

CRP	NO OF PATIENTS
<150 mg/dl	20
>/150 mg/dl	30



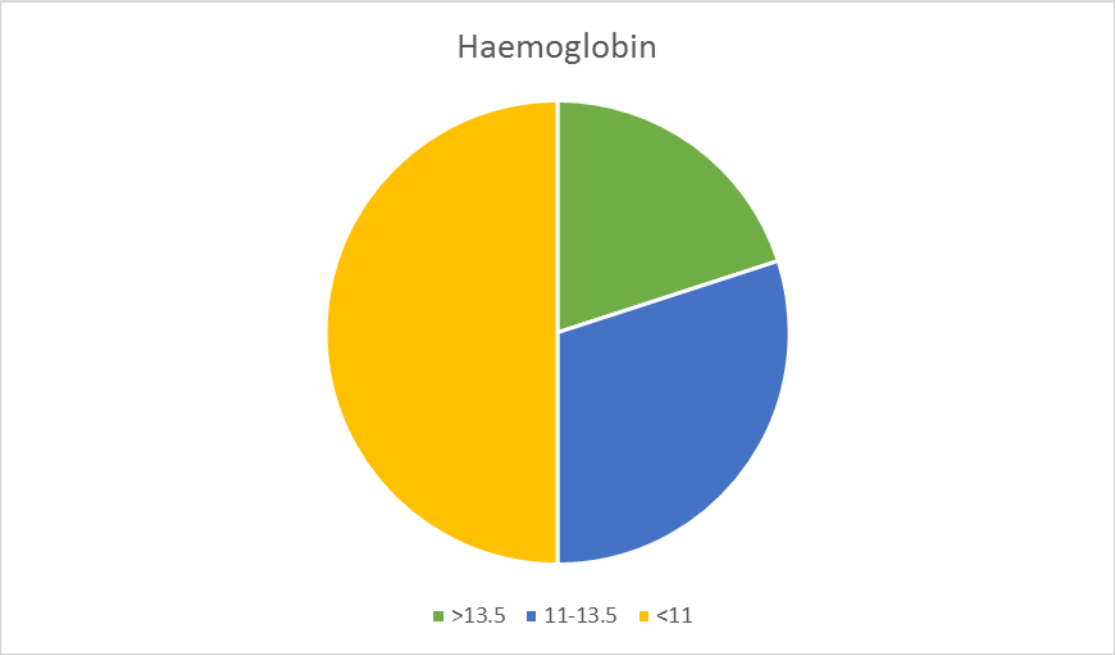
Total Count

TC	NO OF PATIENTS
<15 / cu.mm	20
15-25/ cu.mm	21
>25 / cu.mm	19



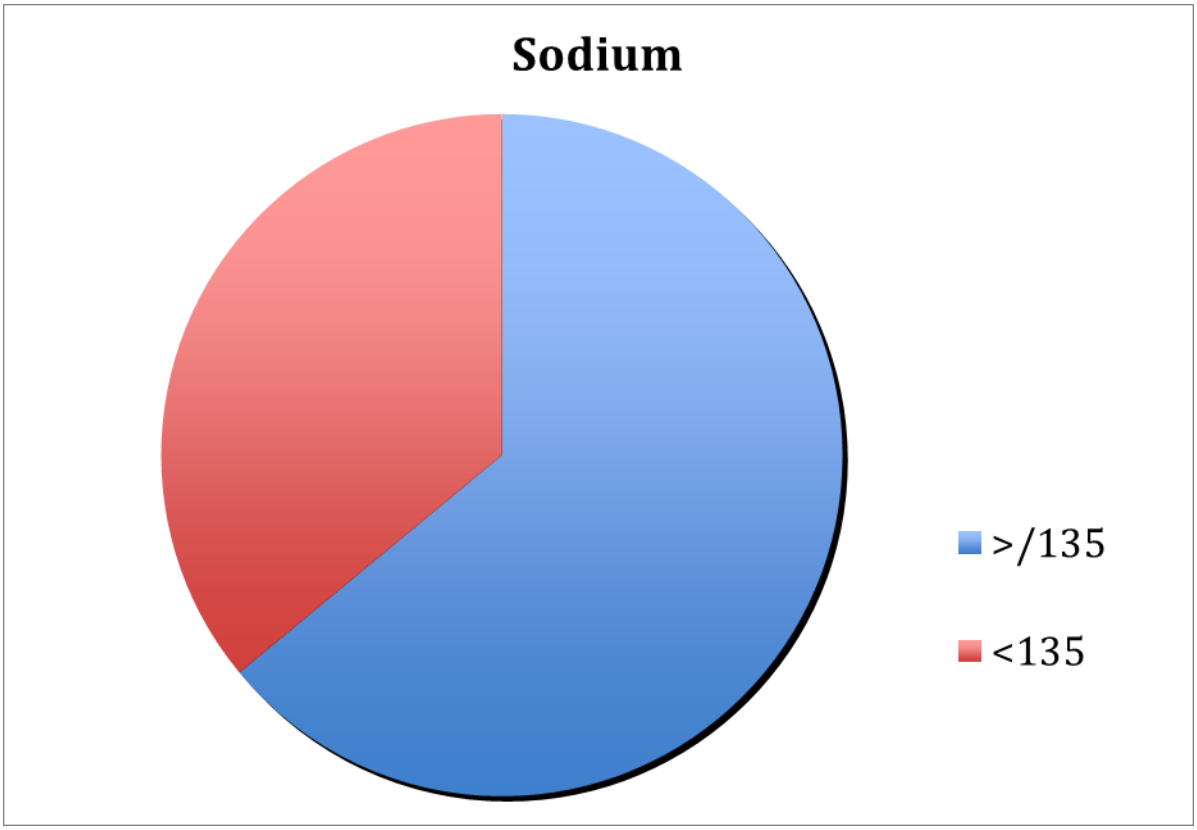
Haemoglobin

HB	NO OF PATIENTS
>13.5 g/dl	10
11-13.5 g/dl	15
<11	25



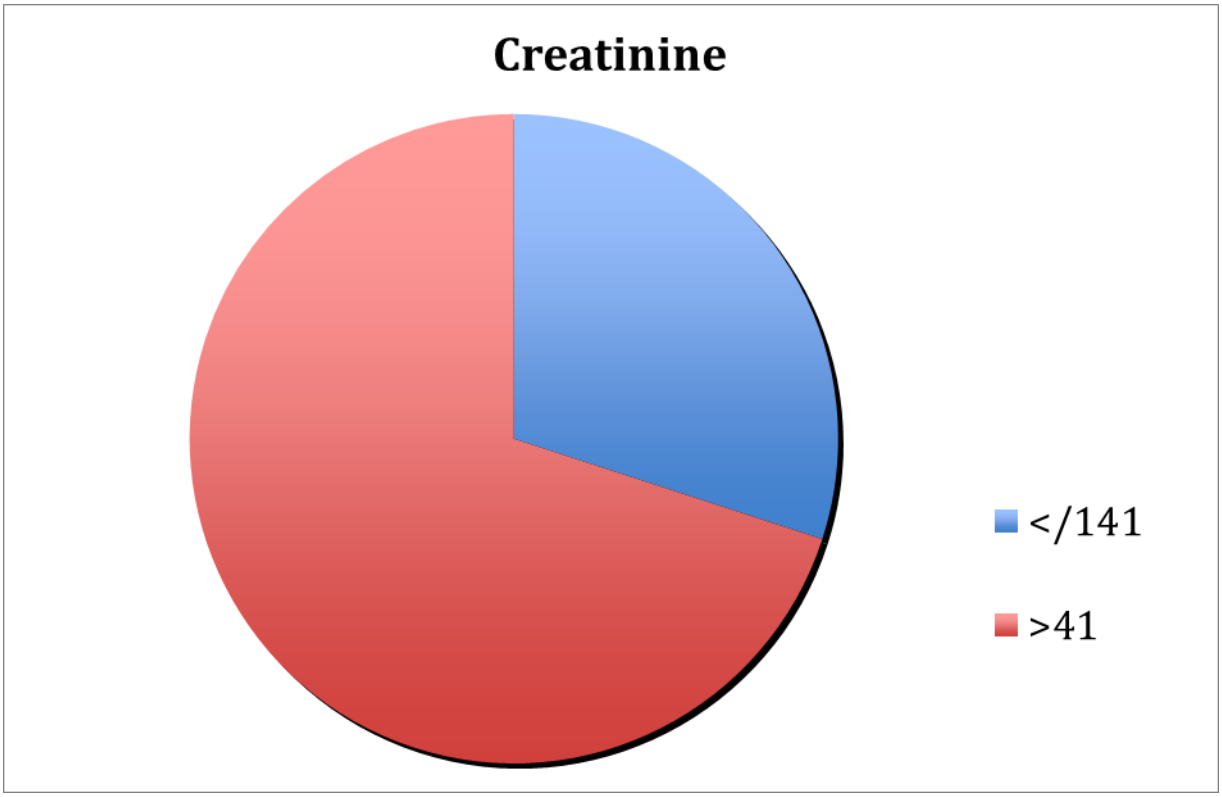
Sodium

Sodium	NO OF PATIENTS
>/135 mmol/L	32
<135 mmol/L	18



Creatinine

Creatinine	NO OF PATIENTS
</ 141 micmol/L	15
>141 micmol/L	35

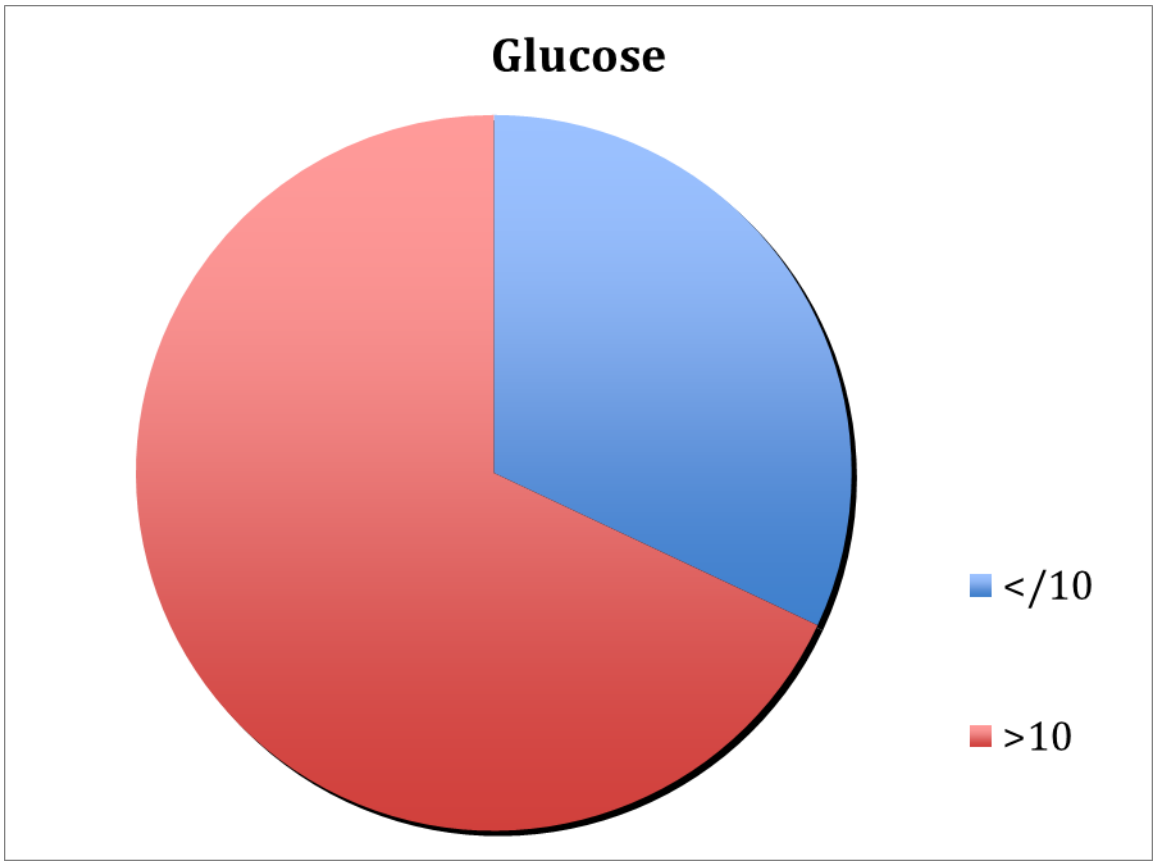


To convert the values of creatinine from mg/dL to micmol/L, divide by 0.01131.

Glucose

Glucose	NO OF PATIENTS
</10 micmol/L	16
>10 micmol/L	34

To convert the values of glucose from mg/dL to micmol/L, divide by 18.015.



LRINEC Score Vs No. of debridements Crosstabulation

		No of debridements		Total
		<3	>/3	
LRINEC Score	<6	15	0	15

	6-7	8	0	8
	>/8	15	12	27
Total		38	12	50

Gram +ve Cocci Vs No of debridements Crosstabulation

		No of debridements		Total
		<3	>/3	
Gram +ve Cocci	-	15	6	21
	+	23	6	29
Total		38	12	50

Gram –ve bacilli Vs No of debridements Crosstabulation

		No of debridements		Total
		<3	>/3	
Gram –ve bacilli	-	18	2	20
	+	20	10	30
Total		38	12	50

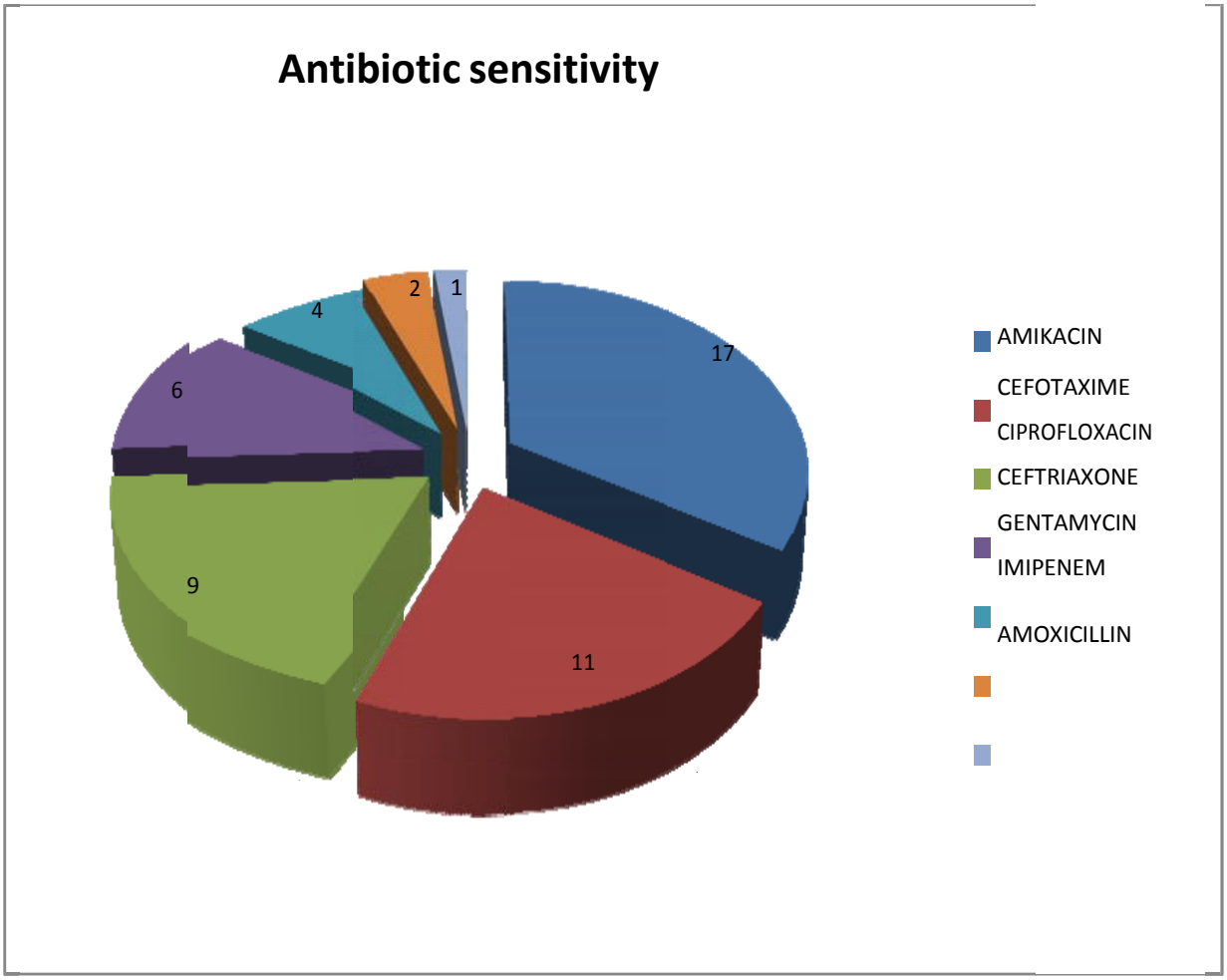
ORGANISMS GROWN ON CULTURE

ORGANISMS	NUMBER OF	PERCENTAGE
Klebsiella	15	30 %
Staphylococcal Positive	10	20 %
E-Coli	7	6 %
Polymicrobial	9	18%
Citrobacter	1	2 %
Proteus	4	8 %
Pseudomonas	7	14 %
Staphylococcal negative	2	4 %

ANTIBIOTIC SENSITIVITY

ANTIBIOTICS	NUMBER OF PATIENTS	PERCENTAGE
AMIKACIN	17	34 %
CEFOTAXIME	11	22 %
CIPROFLOXACIN	9	18 %
CEFTRIAXONE	6	12 %
GENTAMYCIN	4	8 %
IMIPENEM	2	4 %

AMOXICILLIN	1	2 %



LIMB VASCULARITY

As femoral artery in all our patients under study has been found to be unaffected. It's not taken into account.

RIGHT POPLITEAL A VS NO OF DEBRIDEMENTS CROSSTAB

		No of Debridements		Total
		<3	>/3	
Right Popliteal A. pulse	-	1	2	3
	+	37	10	47
Total		38	12	50

RIGHT POSTERIOR TIBIAL A VS NO OF DEBRIDEMENTS CROSSTAB

		No of Debridements		
		<3	>/3	Total
Right Posterior Tibial A. pulse	-	5	5	10
	+	33	7	40
Total		38	12	50

RIGHT DORSALIS PAEDIS A VS NO. OF DEBRIDEMENTS CROSSTAB

		No of Debridements		
		<3	>/3	Total
Right Dorsalis paedis A. pulse	-	15	6	21
	+	23	6	29
Total		38	12	50

LEFT POPLITEAL A VS NO OF DEBRIDEMENTS CROSSTAB

		No of Debridements		Total
		<3	>/3	
Left popliteal A. pulse	-	2	2	4
	+	36	10	46
Total		38	12	50

LEFT POSTERIOR TIBIAL A VS NO OF DEBRIDEMENTS CROSSTAB

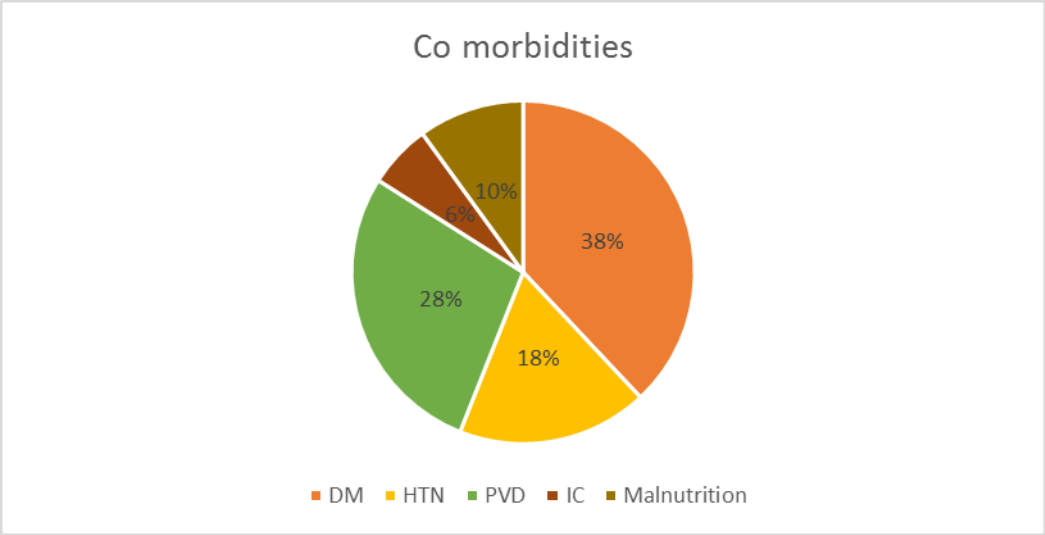
			No of Debridements		Total
			<3	>/3	
Left PTA pulse	-		3	4	7
	+		35	8	43
Total			38	12	50

LEFT DORSALIS PAEDIS VS NO OF DEBRIDEMENTS CROSSTAB

		No of Debridements		Total
		<3	>/3	
Left Dorsalis Pedis A pulse	-	9	8	17
	+	29	4	33
Total		38	12	50

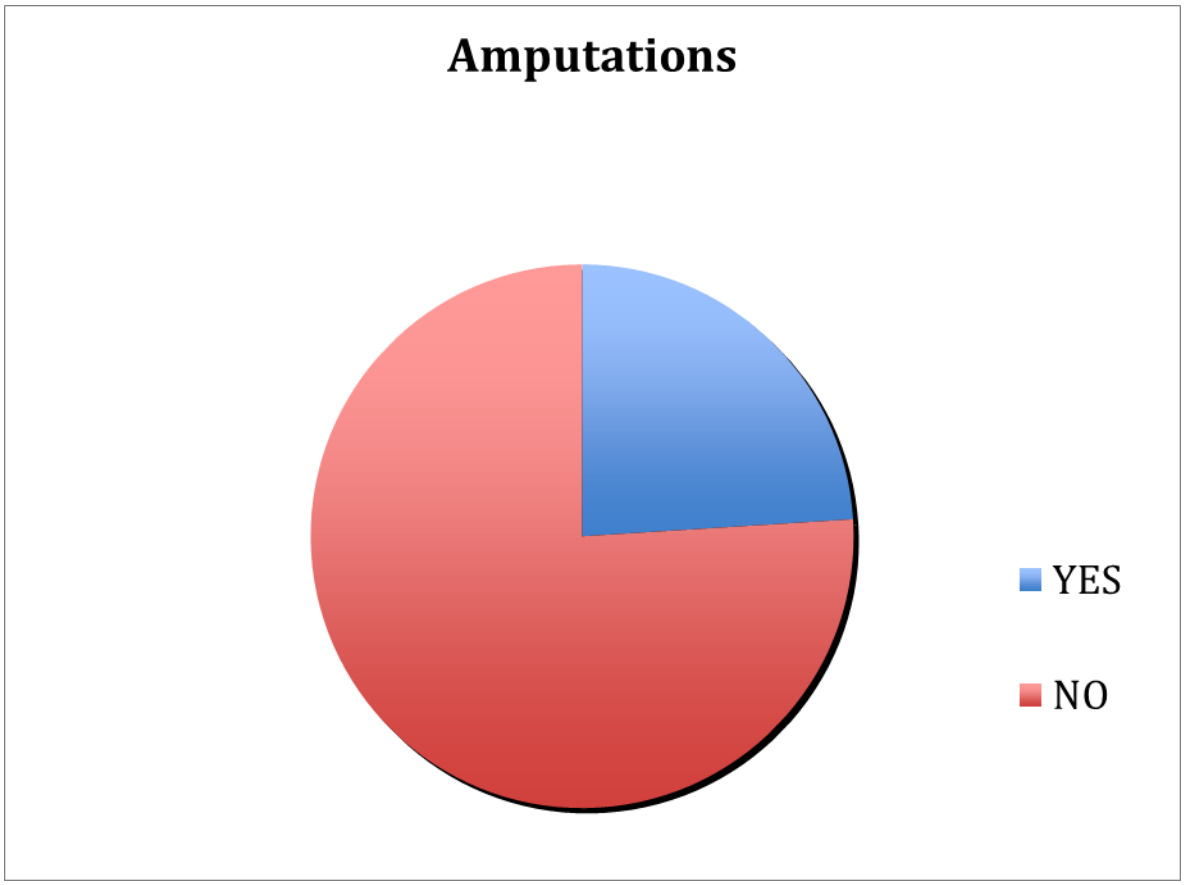
Co Morbidities

Co Morbidities	NO OF PATIENTS
DM	19
PVD	14
HTN	9
Malnutrition	5
Immunocompromised	3



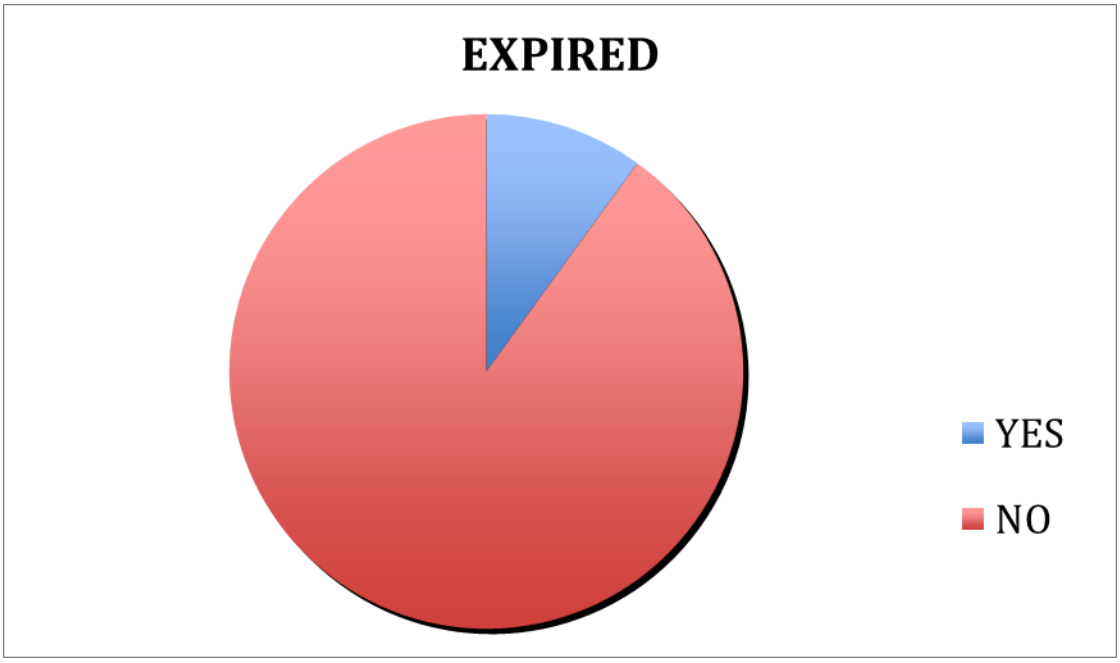
Amputation

Amputation	NO OF PATIENTS
YES	12
NO	38



Death

EXPIRED	NO OF PATIENTS
YES	5
NO	45



DISCUSSION

DISCUSSION

Necrotizing fasciitis was first described as a rapidly spreading gangrene of the subcutaneous tissue caused by beta haemolytic streptococci group A. This disease was later considered as a clinical entity rather than a specific bacterial infection . Many virulent organisms can cause necrotizing fasciitis.[58]

Necrotizing fasciitis is a surgical emergency . Early recognition and prompt aggressive surgical debridement of all necrotic tissue is critical for survival.[59]

Early diagnosis of necrotising fasciitis is very much essential in halting the progression of the disease and for better prognosis. Late detection is almost always associated with a grave prognosis.

We have diagnosed necrotizing fasciitis whenever there is a necrosis of subcutaneous tissues extending through the fascial planes . Paucity of cutaneous findings early in the course of disease makes it difficult to diagnose the condition early . often the disease is masqueraded as cellulitis or abscesses . In these patients diagnosis has been made when the infection progressed despite treatment with broad spectrum intravenous antibiotics.

It has been shown by numerous studies in the past that early recognition and surgical intervention at the earliest is the sole factor in preventing the morbidity and mortality in patients with necrotising fasciitis. The paucity of specific cutaneous signs to distinguish necrotising fasciitis from other soft tissue infections such as cellulitis makes the diagnosis extremely difficult.

So a scoring system which is easy to follow and cost effective with high positive and negative predictive value is required. One such scoring system is the LRINEC scoring system devised by Wong. etal in 2005 .Hence we would like to validate this scoring system in our patients and if found to have similar comparable predictive values, it would prove to be a boon to developing countries like India where the mortality of the disease reported ranges from 7 to 76% and also where there is also constraint for resources.

Table comparing the studies done by other authors

	Sample size	Mean Age	Male : Female	Most common site
Wong et al (2004)	89	56	58:36	Lower Limb
Hefny et al (2007)	11	46	9:2	Lower Limb

Rieger et al (2007)	16	47	9:7	Lower Limb
Present study (2014)	50	51	14:11	Lower Limb

The above study shows a comparision of the present study to a similar study conducted by **Hefny et al (2007) , Wong et al (2004) and Rieger et al(2007)**.

The mean AGE of appearance of necrotizing fasciitis in the present study was 51 years, which is similar to those found in Hefny et al and Rieger et al studies. The mean age of presentation in Rieger et al is 56 years which is slightly higher than the present study .

MALE TO FEMALE RATIO in the present study is approximately 9:7, which is similar to Rieger et al, showing the higher incidence of this condition in males . Higher incidence of NF in males may be due to increased outdoor activities of male like working in fields where they are more prone for minor trauma and snake bites.

The MOST COMMON SITE of involvement in our study is consistent with all 3 studies which is the lower limb.

Comparative analysis

	Mean no. of debridements	Mean duration of hospital stay	Mortality
Wong et al (2004)	2.7	40.6	21.3%
Hefny et al (2007)	2	45	18%
Rieger et al (2007)	5.2	46.6	28.7 %
Present study (2014)	1.94	8.3	10%

MEAN NUMBER OF DEBRIDEMENTS done on patients in the present study was 1.9 times which is comparatively low when compared to all the other studies – Hefney et al (2 times) , Wong et al (2.7 times) and Rieger et al (5.2 times) . Early presentation , early diagnosis and early aggressive surgical debridements has a favorable outcome .

MEAN DURATION OF HOSPITAL STAY in the present study was 8.3 days , which is low compared to all the other studies mentioned above.

In the present study predominant organism was Klebsiella seen in 15 cases (30%) , followed by Staphylococcal aureus coagulase positive seen in 10 cases (20%) . Polymicrobial infection was seen in 9 cases (18%).

Most sensitive antibiotic to the growth in present study was Amikacin in 17 cases (34%) followed by cefotaxime in 11 cases (22%) .

Most common co-morbid condition in this study was diabetes mellitus type 2 (38%) .Diabetes as a co-morbid condition contributed more towards complications associated with NF .

Most common life threatening complication come across in this study was Septicemia (5 cases).

MORTALITY RATE in the present study is 10 % which is comparatively low when compared to Hefny et al (18%) Wong et al(21.3%) and Rieger et al (28.7%) studies. Aggressive resuscitation and early aggressive surgical debridement has been the mainstay of our treatment and has contributed to the low mortality in the present study . Administration of antibiotics plays a supportive role but doesn't replace the extensive excision and removal of the source of sepsis.

Following tables shows correlation between LRINEC score and number of debridements.50 patients in this study were split into three groups with

scores of <6, 6-7 and 8-13 and the results were studied in accordance with chi-square test.

LRINEC SCORE VS NO. OF DEBRIDEMENTS CROSSTABULATION

			No of debridements		Total
			<3	>/3	
LRINEC Score	<6	Count	15	0	15
		% within No of debridements	39.5%	0.0%	30.0%
	6-7	Count	8	0	8
		% within No of debridements	21.1%	0.0%	16.0%
	>/8	Count	15	12	27
		% within No of debridements	39.5%	100.0%	54.0%
Total		Count	38	12	50

% within No of debridements	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided) p value
Pearson Chi-Square	13.450[a]	2	.001
Likelihood Ratio	18.012	2	.000
Linear-by-Linear Association	11.423	1	.001
N of Valid Cases	50		

15 patients with LRINEC score between 0-5 underwent less than 3 debridements.

8 patients with LRINEC score between 6-7 underwent less than 3 debridements.

15 patients with Lrinec score between 8-13 underwent less than 3 debridements and 12 patients underwent 3 or more than 3 debridements.

According to Pearson Chi Square test, with a value of 13.450 and degree of freedom of 2, the p value obtained is 0.001 which is <0.05 and is hence **significant**.

GRAM +VE COCCI VS NO OF DEBRIDEMENTS CROSSTABULATION

			No of debridements		Total
			<3	>/3	
Gram +ve Cocci	-	Count	15	6	21
		% within No of debridements	39.5%	50.0%	42.0%
	+	Count	23	6	29
		% within No of debridements	60.5%	50.0%	58.0%
Total		Count	38	12	50

% within No of debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.415[a]	1	.520
Continuity Correction[b]	.095	1	.758
Likelihood Ratio	.411	1	.521
Fisher's Exact Test			
N of Valid Cases	50		

15 patients with culture negative for Gram positive cocci underwent less than 3 debridements and 6 patients underwent 3 or more than 3 debridements .

23 patients with culture positive for Gram positive cocci underwent less than 3 debridements and 6 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 0.415 and degree of freedom of 1, the p value obtained is 0.520 which is >0.05 and is hence insignificant.

GRAM –VE BACILLI VS NO OF DEBRIDEMENTS CROSSTABULATION

			No of debridements		Total
			<3	>/3	
Gram –ve bacilli	-	Count	18	2	20
		% within No of debridements	47.4%	16.7%	40.0%
	+	Count	20	10	30
		% within No of debridements	52.6%	83.3%	60.0%
Total		Count	38	12	50
		% within No of debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.582[a]	1	.058
Continuity Correction[b]	2.417	1	.120
Likelihood Ratio	3.914	1	.048
Fisher's Exact Test			
N of Valid Cases	50		

18 patients with culture negative for Gram negative bacilli underwent less than 3 debridements and 2 patients underwent 3 or more than 3 debridements .

20 patients with culture positive for Gram negative bacilli underwent less than 3 debridements and 10 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 3.582 and degree of freedom of 1, the p value obtained is 0.58 which is >0.05 and is hence insignificant.

Hence there is no correlation between type of microbial flora and number of debridements done or prognosis of necrotising fasciitis.

LIMB VASCULARITY

RIGHT POPLITEAL A VS NO OF DEBRIDEMENTS CROSSTAB

			No of Debridements		Total
			<3	>/3	
Right Popliteal A	-	Count	1	2	3
		% within No of Debridements	2.6%	16.7%	6.0%
	+	Count	37	10	47
		% within No of Debridements	97.4%	83.3%	94.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.185[a]	1	.074
Continuity Correction[b]	1.183	1	.277
Likelihood Ratio	2.635	1	.105
Fisher's Exact Test			
N of Valid Cases	50		

1 patient with absent right popliteal artery pulse underwent <3 debridement and 2 patients underwent 3 or more than 3 debridements.

37 patients with normal right popliteal artery pulse underwent <3 debridement and 10 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 3.185 and degree of freedom of 1, the p value obtained is 0.074 which is >0.05 and is hence insignificant.

RIGHT POSTERIOR TIBIAL A VS NO OF DEBRIDEMENTS CROSSTAB

			No of Debridements		
			<3	>/3	Total
Right Posterior Tibial A	-	Count	5	5	10
		% within No of Debridements	13.2%	41.7%	20.0%
	+	Count	33	7	40
		% within No of Debridements	86.8%	58.3%	80.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.633[a]	1	.051
Continuity Correction[b]	3.022	1	.082
Likelihood Ratio	4.147	1	.042
Fisher's Exact Test			
N of Valid Cases	50		

5 patients with absent Right posterior tibial artery pulse underwent <3 debridement and 5 patients underwent 3 or more than 3 debridements.

33 patients with normal Right posterior tibial artery pulse underwent <3 debridement and 7 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 4.633 and degree of freedom of 1, the p value obtained is 0.051 which is >0.05 and is hence insignificant.

RIGHT DORSALIS PAEDIS A VS NO. OF DEBRIDEMENTS CROSSTAB

			No of Debridements		Total
			<3	>/3	
Right Dorsalis paedis A	-	Count	15	6	21
		% within No of Debridements	39.5%	50.0%	42.0%
	+	Count	23	6	29
		% within No of Debridements	60.5%	50.0%	58.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.415[a]	1	.520
Continuity Correction[b]	.095	1	.758
Likelihood Ratio	.411	1	.521
Fisher's Exact Test			
N of Valid Cases	50		

15 patients with absent Right Dorsalis paedis Artery pulse underwent <3 debridement and 6 patients underwent 3 or more than 3 debridements.

23 patients with normal Right Dorsalis paedis Artery pulse underwent <3 debridement and 6 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 0.415 and degree of freedom of 1, the p value obtained is 0.052 which is >0.05 and is hence insignificant.

LEFT POPLITEAL A VS NO OF DEBRIDEMENTS CROSSTAB

			No of Debridements		Total
			<3	>/3	
Left popliteal A	-	Count	2	2	4
		% within No of Debridements	5.3%	16.7%	8.0%
	+	Count	36	10	46
		% within No of Debridements	94.7%	83.3%	92.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.611[a]	1	.204
Continuity Correction[b]	.434	1	.510
Likelihood Ratio	1.393	1	.238
Fisher's Exact Test			
N of Valid Cases	50		

2 patient with absent left popliteal artery pulse underwent <3 debridement and 2 patients underwent 3 or more than 3 debridements.

36 patients with normal left popliteal artery pulse underwent <3 debridement and 10 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 1.611 and degree of freedom of 1, the p value obtained is 0.204 which is >0.05 and is hence insignificant.

LEFT POSTERIOR TIBIAL A VS NO OF DEBRIDEMENTS CROSSTAB

			No of Debridements		Total
			<3	>/3	
Left Postr Tibial A	-	Count	3	4	7
		% within No of Debridements	7.9%	33.3%	14.0%
	+	Count	35	8	43
		% within No of Debridements	92.1%	66.7%	86.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.902[a]	1	.057
Continuity Correction[b]	3.017	1	.082
Likelihood Ratio	4.229	1	.040
Fisher's Exact Test			
N of Valid Cases	50		

3 patients with absent Left posterior tibial artery pulse underwent <3 debridement and 4 patients underwent 3 or more than 3 debridements.

35 patients with normal Left posterior tibial artery pulse underwent <3 debridement and 8 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 4.902 and degree of freedom of 1, the p value obtained is 0.057 which is >0.05 and is hence insignificant.

LEFT DORSALIS PAEDIS VS NO OF DEBRIDEMENTS CROSSTAB

			No of Debridements		Total
			<3	>/3	
Left Dorsalis Pedis A	-	Count	9	8	17
		% within No of Debridements	23.7%	66.7%	34.0%
	+	Count	29	4	33
		% within No of Debridements	76.3%	33.3%	66.0%
Total		Count	38	12	50
		% within No of Debridements	100.0%	100.0%	100.0%

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.509[a]	1	.066
Continuity Correction[b]	5.715	1	.017
Likelihood Ratio	7.224	1	.007
Fisher's Exact Test			
N of Valid Cases	50		

9 patients with absent Left Dorsalis paedis Artery pulse underwent <3 debridement and 8 patients underwent 3 or more than 3 debridements.

29 patients with normal Left Dorsalis paedis Artery pulse underwent <3 debridement and 4 patients underwent 3 or more than 3 debridements

According to Pearson Chi Square test, with a value of 7.509 and degree of freedom of 1, the p value obtained is 0.066 which is >0.05 and is hence insignificant.

Hence there is relationship between limb vascularity and number of debridements or prognosis of necrotising fasciitis.

Necrotizing Fasciitis leg before debridement

Necrotizing Fasciitis leg after debridement

Necrotizing Fasciitis leg before debridement

Necroti

zing Fasciitis leg -

debridement

Necrotizing Fasciitis leg before debridement

Necrotizing Fasciitis leg after debridement

Necrotizing Fasciitis leg before debridement

Necrotizing Fasciitis leg after debridement

Below knee amputation stump

Below knee amputation stump rawa area

Below knee amputation stump after secondary closure

Bleb formation in the disease

Extensive involvement of lower limb

Necrotizing Fasciitis before debridement

Necrotizing Fasciitis extending upto muscle layer-post debridement

CONCLUSION

CONCLUSION

1. Necrotizing fasciitis is a surgical emergency , early diagnosis of these rapidly progressing and life threatening infections is a crucial prognostic factor for patient survival and relies on correct interpretation of clinical findings.
2. This study was conducted on 50 randomly selected patients, Youngest in this series was 35 years old and oldest patient was 62 years old , with commonest age group being 50 to 60 years
3. Male to female ratio was approximately 9:7

4. The commonest site of involvement was the lower limbs
5. Commonest co-morbid condition associated with Necrotizing fasciitis was diabetes (38%)
6. The most common bacterial isolate found was Klebsiella (30%) and Polymicrobial infections were seen in 9 cases (18%)
7. Amikacin (34%) was the most sensitive antibiotic in the study
8. 12 patients had to undergo lower limb amputations ranging from forefoot to above knee amputations.
9. Mean duration of hospital stay was 8.3 days
10. Mean number of debridements performed was 1.7 times
11. Mortality rate was 14%

In this study, DOPPLER ULTRASOUND of the affected limb and type of MICROBIAL FLORA have been found not to correlate with the prognosis of necrotising fasciitis and in no way help in determining the outcome of the disease.

But LRINEC SCORING SYSTEM has been found to be a reliable scoring system in early detection of necrotising fasciitis and in predicting the prognosis of the disease. A score more than 8 is almost definitive of necrotizing fasciitis and mandates active debridement and aggressive management for a better outcome.

Hence patients with provisional diagnosis of necrotizing fasciitis should be diagnosed early and treated aggressively with wound debridements as early as possible , if necessary with major amputations and broad spectrum antibiotics in order to bring down the morbidity and mortality.

SUMMARY

SUMMARY

This study shows that:

1. There exists a significant correlation between LRINEC scoring system and prognosis of Necrotizing fasciitis.

2. But there is no significance between the type of MICROBIAL FLORA or VASCULARITY of the affected limb and the outcome of necrotizing fasciitis.

3. Early detection and aggressive debridement are essential in determining the outcome of necrotising fasciitis.

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PATIENT PROFORMA

Name: Age: Sex:

IP No. :

DOA: DOD:

Diagnosis:

Presenting complaints:

Co-morbid illness:

Past surgical /Medical history:

On examination:

General condition:

VITALS:

PR: BP: RR:

CVS:

RS:

P/A:

L/E:

Microbial Flora (Wound c/s) :	
Gram + Cocci	
Gram - Bacilli	

LRINEC Score :

Factors	Score
C - reactive protein, mg/L < 150 - 0 >/ 150 - 4	
Total White Cell Count, per cu. mm <15 - 0 15-25 - 1 >25 - 2	
Haemoglobin, g/dL >13.5 - 0 11-13.5 - 1 <11 - 2	
Sodium, mg/dl >135 - 0 <135 - 2	
Creatinine, micmol/L </141 - 0 >141 – 2	
Glucose, micmol/L </10 - 0 >10 - 1	

Limb Vascularity : Doppler	**Right**	**Left**
Femoral A.		
Popliteal A		
Posterior Tibial A		
Dorsalis Paedis A		

Outcome:

No. of Debridements :

Length of hospital stay:

Amputation:

Mortality:

Condition on discharge:

Key to master chart

Ip No – Inpatient Number

CRP – C Reactive protein

S – Score

TC – Total Count

HB – Haemoglobin

Na – Serum Sodium

Cr – Serum Creatinine

Glc – Glucose

Lrinec S – LRINEC score

FA – Femoral A

PA – Popliteal A

PTA – PostrTibial A

DP – DorsalisPaedis A

G+C – Gram positive cocci

G-B – Gram negative bacilli

Stay – Hospital stay duration

Co Morb – Associated co morbities

DM – Diabetes mellitus

PVD – Peripheral vascular disease

HTN – hypertension

Alc – Alcoholic

IC – Immunocompromised

Mal –Malignancy

No of Deb – Number of debridments